CLAIMS

What is claimed is:

1. A method of ameliorating one or more symptoms of atherosclerosis in a mammal, said method comprising administering to said mammal a phospholipid in an amount sufficient to ameliorate one or more symptoms of atherosclerosis.

2. The method of claim 1, wherein said phospholipid is a phospholipid that inhibits upregulation of an MKP-1 gene.

3. The method of claim 1, wherein said phospholipid is a phospholipid selected from the group consisting of phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and phosphatidyl inositol, and said phospholipid comprises independently selected fatty acids in the *sn*-1 and *sn*-2 positions ranging in length from about 4 to about 24 carbons.

4. The method of claim 3, wherein the fatty acids in in the *sn*-1 and *sn*-2 positions are independently selected from the group consisting of propionoyl, butanoyl, pentanoyl, caproyl, heptanoyl, capryloyl, nonanoyl, capryl, undcanoyl, lauroyl, tridecanoyl, and myristoyl.

5. The method of claim 1, wherein said phospholipid is 1,2-dimyristoyl-*sn*-glycero-3-phosphocholine or an analogue thereof.

6. The method of claim 1, wherein said phospholipid is provided in a unit dose form.

7. The method of claim 1, wherein said phospholipid is provided in a combination of phospholipids.

8. The method of claim 1, where said administration is by a route selected from the group consisting of oral administration, nasal administration, rectal administration, intraperitoneal injection, and intravascular injection, subcutaneous injection, transcutaneous administration, and intramuscular injection.

9. The method of claim 1, wherein said administration is oral administration.

10. The method of claim 1, wherein said administration is an injection.

11. The method of claim 1, wherein said symptoms are in a human patient diagnosed as having or at risk for atherosclerosis.

12. The method of claim 1, wherein said symptoms are in a human patient diagnosed as having atherosclerosis.

13. The method of claim 1, wherein said mammal is also administered a statin.

14. The method of claim 1, wherein said mammal is not administered a synthetic peptide.

15. The method of claim 1, wherein said phospholipid is administered in a formulation lacking other active agents.

16. A method of mitigating or preventing a coronary complication associated with an acute phase response to an inflammation in a mammal, wherein said coronary complication is a symptom of atherosclerosis, said method comprising administering to a mammal having said acute phase response, or at risk for said acute phase response, a phospholipid in an amount sufficient to mitigate or prevent said coronary complication.

17. The method of claim 16, wherein said phospholipid is a phospholipid selected from the group consisting of phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and phosphatidyl inositol, and said phospholipid comprises independently selected fatty acids in the *sn*-1 and *sn*-2 positions ranging in length from about 4 to about 24 carbons.

18. The method of claim 17, wherein the fatty acids in in the *sn*-1 and *sn*-2 positions are independently selected from the group consisting of propionoyl, butanoyl, pentanoyl, caproyl, heptanoyl, capryloyl, nonanoyl, capryl, undcanoyl, lauroyl, tridecanoyl, and myristoyl.

19. The method of claim 16, wherein said phospholipid is 1,2-dimyristoyl-*sn*-glycero-3-phosphocholine or an analogue thereof.

20. The method of claim 16, wherein said phospholipid is provided in a unit dose form.

21. The method of claim 16, wherein said phospholipid is provided in a combination of phospholipids.

22. The method of claim 16, where said administration is by a route selected from the group consisting of oral administration, nasal administration, rectal administration, intraperitoneal injection, and intravascular injection, subcutaneous injection, transcutaneous administration, and intramuscular injection.

23. The method of claim 16, wherein said administration is oral administration.

24. The method of claim 16, wherein said administration is an injection.

25. The method of claim 16, wherein said acute phase response is an inflammatory response associated with a recurrent inflammatory disease.

26. The method of claim 16, wherein said acute phase response is associated with a disease selected from the group consisting of leprosy, tuberculosis, systemic lupus erythematosus, polymyalgia rheumatica, polyarteritis nodosa, scleroderma, idiopathic pulmonary fibrosis, chronic obstructive pulmonary disease, Alzheimers Disease AIDS, coronary calcification, calcific aortic stenosis, osteoporosis, and rheumatoid arthritis.

27. The method of claim 16, wherein said acute phase response is an inflammatory response associated with a condition selected from the group consisting of a bacterial infection, a viral infection, a fungal infection, an organ transplant, a wound, an implanted prosthesis, parasitic infection, sepsis, endotoxic shock syndrome, and biofilm formation.

28. A method of mitigating or preventing a coronary complication associated with an acute phase response to an inflammation in a mammal, wherein said coronary complication is a symptom of atherosclerosis, said method comprising:

assaying said mammal for an acute phase protein (APP) level indicative of an acute phase response or a significant risk of an acute phase response; and

administering to a mammal showing an acute phase protein (APP) level indicative of an acute phase response a phospholipid in an amount sufficient to mitigate or prevent said coronary complication.

29. The method of claim 28, wherein said phospholipid is a phospholipid selected from the group consisting of phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and phosphatidyl inositol, and said phospholipid comprises independently selected fatty acids in the *sn*-1 and *sn*-2 positions ranging in length from about 4 to about 24 carbons.

30. The method of claim 29, wherein the fatty acids in in the *sn*-1 and *sn*-2 positions are independently selected from the group consisting of propionoyl, butanoyl, pentanoyl, caproyl, heptanoyl, capryloyl, nonanoyl, capryl, undcanoyl, lauroyl, tridecanoyl, and myristoyl.

31. The method of claim 29, wherein said phospholipid is 1,2-dimyristoyl-*sn*-glycero-3-phosphocholine or an analogue thereof.

32. The method of claim 28, wherein said acute phase response is an inflammatory response associated with a recurrent inflammatory disease.

33. The method of claim 28, wherein said acute phase response is associated with a disease selected from the group consisting of leprosy, tuberculosis, systemic lupus erythematosus, polymyalgia rheumatica, polyarteritis nodosa, scleroderma, idiopathic pulmonary fibrosis, chronic obstructive pulmonary disease, Alzheimers Disease AIDS, coronary calcification, calcific aortic stenosis, osteoporosis, and rheumatoid arthritis.

34. The method of claim 28, wherein said acute phase response is an inflammatory response associated with a condition selected from the group consisting of a bacterial infection, a viral infection, a fungal infection, an organ transplant, a wound, an implanted prosthesis, parasitic infection, sepsis, endotoxic shock syndrome, and biofilm formation.

35. The method of claim 28, wherein said acute phase protein (APP) is a positive APR selected from the group consisting of serum amyloid A, c-reactive protein, serum amyloid P component, C2 complement protein, C3 complement protein, C4 complement protein, C5 complement protein, C9 complement protein, B complement protein, C1 inhibitor, C4 binding protein, fibrinogen, von Willebrand factor, α1-antitrypsin, α1-antichymotrypsin, α2 antiplasmin, heparin cofactor II, plasminogen activator inhibitor I, haptoglobin, haemopexin, ceruloplasmin , manganese superoxide dismutase, α1-acid glycoprotein, haeme oxygenase, mannose binding protein, leukocyte protein I, lipoprotein (a), and lipopolysaccharide binding protein.

36. The method of claim 28, wherein said acute phase protein (APP) is a negative APR selected from the group consisting of albumin, prealbumin, transferin, apoAI, apoAII, α2-HS glycoprotein, inter-α-trypsin inhibitor, histidine rich glycoprotein.

37. The method of claim 28, wherein said phospholipid is provided in a unit dose form.

38. The method of claim 28, wherein said phospholipid is provided in a combination of phospholipids.

39. The method of claim 28, where said administration is by a route selected from the group consisting of oral administration, nasal administration, rectal administration, intraperitoneal injection, and intravascular injection, subcutaneous injection, transcutaneous administration, and intramuscular injection.

40. The method of claim 28, wherein said administration is oral administration.

41. The method of claim 28, wherein said administration is an injection.

42. A method of inhibiting a symptom of an inflammatory condition, said method comprising administering to a mammal exhibiting a symptom of a pathology characterized by an inflammatory response a phospholipid in an amount sufficient to mitigate a symptom associated with said inflammatory condition.

43. The method of claim 42, wherein said phospholipid is a phospholipid selected from the group consisting of phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and phosphatidyl inositol, and said phospholipid comprises independently selected fatty acids in the *sn*-1 and *sn*-2 positions ranging in length from about 4 to about 24 carbons.

44. The method of claim 43, wherein the fatty acids in the *sn*-1 and *sn*-2 positions are independently selected from the group consisting of propionoyl, butanoyl, pentanoyl, caproyl, heptanoyl, capryloyl, nonanoyl, capryl, undcanoyl, lauroyl, tridecanoyl, and myristoyl.

45. The method of claim 42, wherein said phospholipid is 1,2-dimyristoyl-*sn*-glycero-3-phosphocholine or an analogue thereof.

46. The method of claim 42, wherein said phospholipid is provided in a unit dose form.

47. The method of claim 42, wherein said phospholipid is provided in a combination of phospholipids.

48. The method of claim 42, where said administration is by a route selected from the group consisting of oral administration, nasal administration, rectal administration, intraperitoneal injection, and intravascular injection, subcutaneous injection, transcutaneous administration, and intramuscular injection.

49. The method of claim 42, wherein said symptoms are in a human patient diagnosed as having or at risk for atherosclerosis.

50. The method of claim 42, wherein said symptoms are in a human patient diagnosed as having atherosclerosis.

51. The method of claim 52, wherein said inflammatory condition is selected from the group consisting of rheumatoid arthritis, lupus erythematous, polyarteritis nodosa, osteoporosis, and a viral illness.

52. A composition for ameliorating one or more symptoms of atherosclerosis, said composition comprising a phospholipid in a unit dose form.

53. The composition of claim 52, wherein said unit dose form further comprises a pharmacologically acceptable excipient.

54. The composition of claim 52, wherein said phospholipid is a phospholipid selected from the group consisting of phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and phosphatidyl inositol, and said phospholipid comprises independently selected fatty acids in the *sn*-1 and *sn*-2 positions ranging in length from about 4 to about 24 carbons.

55. The composition of claim 54, wherein the fatty acids in the *sn*-1 and *sn*-2 positions are independently selected from the group consisting of propionoyl, butanoyl, pentanoyl, caproyl, heptanoyl, capryloyl, nonanoyl, capryl, undcanoyl, lauroyl, tridecanoyl, and myristoyl.

56. The composition of claim 52, wherein said phospholipid is 1,2-dimyristoyl-*sn*-glycero-3-phosphocholine or an analogue thereof.

57. A kit for ameliorating one or more symptoms of atherosclerosis, said kit comprising:

a phospholipid; and

instructional materials teaching the administration of said phospholipid to a mammal to mitigate one or more symptoms of atherosclerosis.

58. The kit of claim 57, wherein said phospholipid is a phospholipid selected from the group consisting of phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and phosphatidyl inositol, and said phospholipid comprises independently selected fatty acids in the *sn*-1 and *sn*-2 positions ranging in length from about 4 to about 24 carbons.

59. The kit of claim 58, wherein the fatty acids in the *sn*-1 and *sn*-2 positions are independently selected from the group consisting of propionoyl, butanoyl, pentanoyl, caproyl, heptanoyl, capryloyl, nonanoyl, capryl, undcanoyl, lauroyl, tridecanoyl, and myristoyl.

60. The kit of claim 57, wherein said phospholipid is 1,2-dimyristoyl-*sn*-glycero-3-phosphocholine or an analogue thereof.

61. The kit of claim 57, wherein said phospholipid is in a unit dose form.

62. A kit for inhibiting a symptom of an inflammatory condition, said kit comprising:

a phospholipid; and

instructional materials teaching the administration of said phospholipid to a mammal to mitigate one or more symptoms of an inflammatory condition.

63. The kit of claim 66, wherein said phospholipid is a phospholipid selected from the group consisting of phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and phosphatidyl inositol, and said phospholipid comprises independently selected fatty acids in the *sn*-1 and *sn*-2 positions ranging in length from about 4 to about 24 carbons.

64. The kit of claim 63, wherein the fatty acids in the *sn*-1 and *sn*-2 positions are independently selected from the group consisting of propionoyl, butanoyl, pentanoyl, caproyl, heptanoyl, capryloyl, nonanoyl, capryl, undcanoyl, lauroyl, tridecanoyl, and myristoyl.

65. The kit of claim 66, wherein said phospholipid is 1,2-dimyristoyl-*sn*-glycero-3-phosphocholine or an analogue thereof.

66. The kit of claim 66, wherein said phospholipid is in a unit dose form.

67. A method of repairing tissue damage associated with atherosclerosis, said method comprising administering to said mammal a phospholipid in an amount sufficient to partially or fully repair tissue damage to a tissue wherein said damage is tissue damage associated with atherosclerosis.

68. The method of claim 67, wherein said phospholipid is a phospholipid that inhibits upregulation of an MKP-1 gene.

69. The method of claim 67, wherein said phospholipid is a phospholipid selected from the group consisting of phosphatidyl choline, phosphatidyl serine, phosphatidyl ethanolamine, and phosphatidyl inositol, and said phospholipid comprises

independently selected fatty acids in the sn-1 and sn-2 positions ranging in length from about 4 to about 24 carbons.

70. The method of claim 69, wherein the fatty acids in in the sn-1 and sn-2 positions are independently selected from the group consisting of propionoyl, butanoyl, pentanoyl, caproyl, heptanoyl, capryloyl, nonanoyl, capryl, undcanoyl, lauroyl, tridecanoyl, and myristoyl.

71. The method of claim 67, wherein said phospholipid is 1,2-dimyristoyl-sn-glycero-3-phosphocholine or an analogue thereof.

72. The method of claim 67, wherein said phospholipid is provided in a unit dose form.

73. The method of claim 67, wherein said phospholipid is provided in a combination of phospholipids.

74. The method of claim 67, where said administration is by a route selected from the group consisting of oral administration, nasal administration, rectal administration, intraperitoneal injection, and intravascular injection, subcutaneous injection, transcutaneous administration, and intramuscular injection.

75. The method of claim 67, wherein said administration is oral administration.

76. The method of claim 67, wherein said administration is an injection.

77. The method of claim 67, wherein said symptoms are in a human patient diagnosed as having or at risk for atherosclerosis.

78. The method of claim 67, wherein said symptoms are in a human patient diagnosed as having atherosclerosis.

79. The method of claim 67, wherein said mammal is also administered a statin.

80. The method of claim 67, wherein said mammal is not administered a synthetic peptide.

81. The method of claim 67, wherein said phospholipids is administred in a formulation lacking other active agents.

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